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FINAL REPORT - A BIBLIOGRAPHY OF PUBLISHED DATA ON STUDIES OF ACUTE TOXICITY, SUBLETHAL EFFECTS AND SECONDARY TOXICITY ON WILDLIFE SPECIES

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The Illinois Department of Conservation
Division of Wildlife Resources

EXECUTIVE SUMMARY

This final report completes a contract for the period July 1, 1986 to June 1, 1987 to provide "a bibliography of published data on field studies of acute toxicity, sublethal effects and secondary toxicity on wildlife species". An interim report was submitted September 1, 1986 that listed 186 publications pertaining to organophosphorus (OP) compounds and their metabolites that may have direct toxic effects on non-target wildlife and/or secondary effects on prey/scavenger species, especially raptors. The methods section of that report detailed how the literature search was conducted and indexing services used.

For this final report, additional references were added, and the resulting 210 citations were arranged for reader convenience in 3 general groupings: General Bibliography, Fenthion and Related Phosphorothionates, and

Secondary Toxicity and Hazard Assessment. In addition, to provide a brief overview of pertinent topics, key selected references were reviewed and their contents paraphrased under the headings History, Structure, Mode of Action, Factors Affecting Toxicity, Clinical Signs, Side Effects, Fenthion, and Secondary Toxicity and Hazard Assessment.

It is apparent from this review that the subject of secondary toxicity to wildlife species has been poorly researched. However, enough instances have been documented throughout the world to indicate there is clear risk to non-target species. While OP's and related compounds have proven to be useful and beneficial to agricultural interests, their potential sublethal and secondary toxic effects on wildlife warrant public concern. In the absence of more definitive research on population effects, it would be judicious to err on the side of environmental quality and wildlife health when weighing cost-benefits of specific applications. When use of OP's are clearly in the public interest it should not be precluded; however, hazard should be assessed prior to each application and strategies developed to eliminate or minimize risk. When unavoidable risk is posed, measures for mitigation should be incorporated in regulatory guidelines.

HISTORY[1]

In addition to being an essential component of protoplasm, organophosphorus (OP) compounds have been artificially synthesized for use as lubricants, oil additives, plasticizers, and pesticides. The toxic effects of OP compounds were first discovered in 1932. During World War II, phosphorus compound research was pursued in the development of nerve poisons and contact insecticides. The discovery of parathion in 1944 and its structural-activity relationship opened the door for development of additional compounds. By slight structural modifications many more, often less toxic, insecticides were synthesized as well as acaricides, plant and animal systemic insecticides, nematocides, insect chemosterilants, fungicides, herbicides, plant growth regulators, rodenticides, insecticide synergists, and insect repellents. The first practical insecticide, Baldan (containing TEPP), was marketed in Germany in 1944. By 1974, more than 140 pesticides of organophosphorus origin were in practical use (see appendix 1 for a condensed summary) and more than 60,000 tons of these pesticides were produced in the United States alone.

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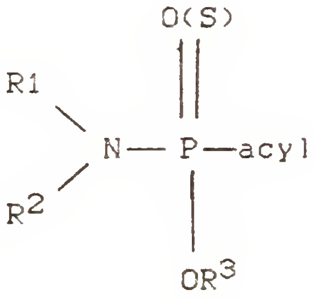
[1] This summary has been condensed from Buck et al. (1973) and Eto (1974).

STRUCTURE

Phosphorus is a highly reactive element, yielding pentavalent or higher compounds. Some organic phosphorus compounds are essential to life being found in nucleic acids, nucleotide coenzymes, metabolic intermediates and phoshatids. Variation in the groups attached to phosphorus's 4 binding sites is almost limitless, and compounds displaying a wide variety of biological activities are possible. The general formula of an OP compound with contact insecticide properties is:



where R^1, R^2, and R^3 are alkyl groups and "acyl" is an inorganic or organic acid radical such as CL, F, SCN, and CH_3COO. Currently, most OP pesticides are neutral esters or amides derived from phosphoric acid, it's anhydride, or sulfur analogs; others are fluorides, or recently phosphonic acid or its derivities. One class of OP compounds, the thionophosphates, (parathion for example) must undergo microsomal oxidation before it can produce toxic effect. Carbamate compounds contain cyclic or aliphatic derivatives

of carbamic acid, and unlike organophosphorus pesticides produce reversible enzyme inhibition.

MODE OF ACTION

OP and carbamate insecticides exert their toxic effects by inhibition of acetylcholinesterase (AChE). This enzyme, critical to nerve function, hydrolyzes the nerve impulse transmitter, acetylcholine, into acetic acid and choline by removing the hydroxyl ion from serine at the active site. During nerve simulation acetylcholine is released and must be rapidly hydrolized before a second impulse arrives. In insects the neuromuscular junctions are not cholinergic but acetylcholine is the transmitter in the synapses of the central nervous system, a chain of ventral ganglia. The disruption of acetylcholine hydrolyses can be severe or lethal. In vertebrates, the synapses of the central nervous system, neuromuscular junctions of motor nerves, postganglionic parasympathetic nerve terminals and sympathetic terminals on sweat glands, blood vessels and adrenal medulla are all cholinergic. Lethal OP poisoning in mammals is usually due to respiratory paralysis. Evidence indicates that organisms can develop tolerance to repeated sublethal doses; the cholinergic receptors becoming refractory to repeated exposure.

FACTORS AFFECTING TOXICITY

Toxicity can vary depending upon the nature of the chemical and the species involved. Other factors affecting the efficacy of OP pesticides include penetration, metabolism, storage and excretion, transport to target, nature of target, and interaction factors. Chemicals may enter an organism through the digestive tract, skin or cuticle, spiracle, mucous membranes, leaves, stem or roots. Dose levels may have to be adjusted to accommodate different routes of application. For example, a much higher dose of fenthion must be used when it is applied as a topical avicide than would be necessary to produce lethal effect if given orally.

Selectivity is often based on an organism's ability to detoxify a given pesticide. For example, malathion is highly toxic to most insects and less toxic to mammals. Mammalian liver carboxyesterase is about 2,500 times as insensitive to inhibition as the insect enzyme. This provides mammals on opportunity to detoxify malathion.

Esterases in the liver and serum can detoxify some OP pesticides and some of these pesticides can inhibit esterase activity before there is significant depression of AChE activity. By this mechanism some pesticides may potentiate others. Because these esters also metabolize drugs, animals

suffering pesticide poisoning may also have increased sensitivity to certain drugs, such as succinylcholine.

Animals differ in their ability to store and excrete OP pesticides. Soluble OP compounds can be excreted from the vertebrate body in the urine, or with bile into the feces. Mice were able to excrete Salithion so rapidly that only 2.4 % of the administered dose was present 3 hrs after the oral dosing as opposed to the 4 % of the applied dose found in houseflies 24 hrs after tropical treatment. The relatively low chronic toxicity of OP compounds in vetebrates is due to their clearance abilities. Storage of toxins may also effect tolerance. The female cockroach has greater body fat than the male and is twice as tolerant to schradan as the male due to her increased fat-storage capacity.

Toxin selectivity may also be due to factors that affect transport to the reaction site. For example, tetrachlorvinphos has poor solubility in both water and organic solvents and thus slow penetration and transport to target areas. These properties contribute to its low mammalian toxicity making it useful in dairies and against garden pests.

The nature of the target can affect selectivity toxicity. Mammalian AChE, for example, recovers rapidly from phosphorylation by Haloxon whereas that of parasitic nematodes does not recover.

In mice famphur is 90% degraded in 1 hr but only 10% degraded in 1 hr in milkweed bugs. However, the toxicity is relatively the same for both organisms. The discrepancy is due to the higher sensitivity of mouse AChE (32 times) to the oxon analog of famphur than that of the milkweed bug. Thus toxicity can be affected by an interaction of factors and often selectivity can not be attributed to a sole factor.

CLINICAL SIGNS

Most of the clinical signs of OP and carbamate pesticide poisoning are due to the overstimulation of the parasympathic nervous system. The signs are non-specific and include, profuse salivation, abdominal cramps, vomiting, diarrhea, excessive lacrimation, sweating, shortness of breath, constriction of the pupil, pallor, and incontinence of urine and feces. Excessive secretion of the respiratory tract may cause coughing. There may also be excessive stimulation of the skeletal muscles causing twitching of the various muscle groups and often generalized tetany causing the animal to walk in a stiffed legged fashion. This may progress into weakness and paralysis of skeletal muscles. Signs of central nervous system involvement differ depending upon the species involved and can include hyperactivity, depression, and convulsive seizures. Death usually occurs due to hypoxia caused by bronchoconstriction; excessive

respiratory tract secretions in the bronchi, and erratic, slowed heartbeat. Not all animals will show all to the signs, however, if several animals are poisoned most reported clinical signs will be present in some of the animals. Poisoning by systemic OP pesticides produces more confusing clinical signs because these pesticides frequently contain chlorinated hydrocarbons which produce different toxic effects. Systemic poisoning may not be apparent until 12 to 36 hrs after exposure.

SIDE EFFECTS

OP compounds are known to produce toxic side effects not related to AChE inhibition in the nervous system. Several OP compounds can cause delayed neurotoxicity; a special chronic neurotoxicity caused by an unknown mechanism. This condition is accompanied by irreversible demyelination in the central and peripheral neverous system in certain species of vertebrates. OP compounds may also be teratogenic. Although previous safety evaluations have been conducted in vivo, they may now be conveniently performed in cell culture. OP compounds may also effect enzymes other than cholinesterases. The fungicide Kitazin for example, inhibits the incorporation of glucosamine in the fungus mycelia cell wall. Commonly applied to plants, a desired characteristic of organophosphorus pesticide is nonphytotoxicity. However, some pesticides may also effect

plant metabolism; for example, some OP insecticides disturb coloration in apples.

Fenthion

Fenthion was originally developed in 1958 by Bayer AG as a general purpose insecticide with systemic action, particularly effective against flies and mosquitoes. It is a highly persistent pesticide being relatively stable to acid and alkaline hydolysis and heat. Oxidative desulfuration of fenthion can proceed in plants, animals or sunlight producing sulfoxide and sulfone. These metabolites of fenthion are also insecticidal.

Fenthion, mevinphos, and parathion have been used as avicides on large concentrations of blackbirds, starlings and quelea. They have been applied as treated baits, dermal contact sprays, treated water, and wicked perches. Bird mortality from dermal exposure usually occurs in 3 to 12 hours but can take days. Treated birds and contaminated insects have been responsible for secondary poisoning of insectiverous birds and raptors. Although OPs are not considered persistent pesticides, the inhibition of AChE they cause is irreversible. The effects of OP poisoning are cumulative and reversed only by the synthesis of new AChE. The availability of contaminated prey from avian pest control operations is sufficient to cause the loss of many

predators; however, the extent of secondary poisoning has not been well researched.

Fenthion and famphur are also used as dermal insecticides in livestock operations. These OPs are known to persist on the hair of treated animals for as long a 3 months. The scavenged carcasses of treated animals have been the source of unintentional and secondary poisoning of raptors and other birds.

SECONDARY TOXICITY/HAZARD ASSESSMENT[2]

Grue et al. (1983) reported 418 instances of wildlife mortality worldwide resulting from application of OP's for agricultural pest control. Poisoned animals represented 28 families of birds and 5 mammal families. Unpublished data from USDA-AIDS and FWS sources indicated numerous other undocumented instances of wildlife mortality suggesting the published record represents a small fraction of non-target wildlife deaths attributable to OP applications. Two unpublished, but documented instances of raptors dying following avicide formulations of fenthion to control large aggregations of mixed blackbirds in Illinois (Illinois Dept. Cons. unpubl. data) clearly illustrates the potential for secondary toxicity associated with OP use.

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[2] This summary has been condensed from Bruggers et al. (1986), Buck et al. (1973), and Grue et al. (1983).

In the late 1970's, the FWS Patuxent Wildlife Research Center began to test all birds received for possible OP poisoning. They have reported 5 instances of raptors poisoned by famphur and fenthion. In each case, the deaths resulted from OP insecticides poured on the backs of livestock to control ectoparasites. In one instance, an eagle was poisoned after feeding on a cow carcass that had been treated with famphur 3 months earlier. Clearly, the persistence of these pesticides (and/or their sometimes toxic metabolites) is greater than previously thought and new recommendations about application and carcass disposal are needed.

In North America, waterfowl, especially geese, were the group most frequently reported by Grue et al. (1983) to be accidently poisoned. This may in part be due to their gregarious behavior and high visibility as well as their feeding habits. Raptors which may be easily attracted to dying animals suffering from OP poisoning are especially vulnerable to secondary poisoning. In contrast, there is a relatively low reported incidence of mammals dying from OP's. Acute toxicity trials indicate that mammals are less susceptible to OP poisoning than are birds. However, the low reporting rate may also be attributable to more secretive behavior and lower visibility.

Widespread use of fenthion and related compounds for quelea control in Africa provides some measure of risk of secondary toxicity to raptors and other predators/ scavengers. For example, an unpublished study by the USFWS presents strong evidence that predatory birds appear to be attracted to areas treated with OP's. Target species treated at one site and dying elsewhere because of foraging habits, movements, etc. considerably extend the area of risk. During quelea control in Africa, debilitated adults and young continued to die up to 7 days post-treatment and were found in an area at least 35 km^2 around the treated roost. One investigator reported quelea dying up to 19 days after one control operation.

Hazard Assessment

It is important to distinguish between hazard and toxicity. Hazard is defined as the likelihood of poisoning occurring under certain conditions and the likelihood of exposure. For example, when fenthion is applied to flat bars and placed on a pigeon roost in and around buildings it is both a high hazard and a toxicant to pigeons. However, due to their behavior (they prefer to perch on other surfaces), fenthion on bars is practically no hazard to song birds. Just because a substance is highly toxic does not necessarily mean it is a great hazard.

Route of Exposure

OP pesticides are applied by a variety of methods that make them more or less available to non-target species. Granivorous birds for example may be poisoned by consuming treated seeds. Even seeds that are covered and germinated may present a hazard to birds such as geese that uproot some of the vegetation they consume. Residues on treated cultivated plants can present a hazard; but it is also important to assess the use of adjacent vegetation if it is likely to become contaminated during pesticide treatment of fieldcrop. Pesticides applied in granular form may be picked up as grit, seeds or accidently with food by granivorous birds. Vegetation and invertebrates in the treated area may also be contaminated when chemicals are washed off the granules.

Secondary poisoning may occur when non-target animals feed on insects or other pest killed or weakened by pesticide treatment. Predators may change their normal food habits to take advantage of a ready food supply. Poisoned non-target animals may themselves become sources of secondary poisoning. It may also be possible to significantly reduce the food supply of certain animals by pesticide application.

Animals may also be poisoned by dermal and inhalation route especially during aerial application of pesticides.

Petroleum products are often used a carriers of OP pesticides. These may stick to the fur or feathers of non-target species and allow the transfer of pesticides to an animals nest or other areas.

The toxic affect of OP compounds could depend on whether the exposure is chronic or acute. Although organophosphorus pesticides are relatively short-lived compared to organochlorides they may persist in soil, water, foliage, invertebrates, and vertebrates for a period of days to several weeks.

Factors Affecting An Organisms Physiological response To OP Pesticides

Physiological and environmental factors can affect the response of organisms to pesticides (Table 1). Although there are many compounding factors and it would be difficult to a priori assess the impact of all of these, it is important to keep these in mind when trying to determine risk assessment and in diagnosis of possible OP pesticide caused mortality.

In general birds are more sensitive to OP pesticides than mammals and mammals are more sensitive than amphibians. Mammalian females tend to be more sensitive than males and young animals more sensitive than old.

Table 1. Factors that can alter the response of animals to toxicants.[1]

PHYSIOLOGICAL FACTORS
Genus
Species
Strain
Breed
Age
Sex
Maturity
Estrus cycle
Pregnancy
Lactation

ENVIRONMENTAL
Season
Temperature
Transpiration
Humidity
Atmospheric contaminants
Air circulation
Light intensity
Light spectrum
Light/dark cycle

WATER
Quality
Quantity
Mode of delivering

DIET
Constituents
Protein level
Quantity
Quality
Contaminants

CAGING
Size
Material
Shape

BEDDING
Type
Quantity

ANIMAL HANDLING
Physical contact
Noise
Commotion
Temperament of handler

HEALTH
Deficiencies
Immunity
Latent infections
Clinical Illness

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[1] from Buck, W. B., G. D. Osweiler, and G. A. VanGelder. 1973. Clinical and diagnostic veterinary toxicology. Kendall/Hunt Publishing Company, Dubuque, Iowa. 380 pp.

Sublethal Effects

OP pesticides may have a number of sublethal effects on non-target species. These may affect not only the individual organism but populations. OP pesticides cause loss of body weight, and hormonal and behavioral changes that can affect reproductive success. Survival of an organism may be impaired due to hypothermia or nervous and/or muscle system changes that affect sensory perception, behavior and physical response to stress and predators. Sublethal effects are more difficult to document but represent the potential for significant losses and may be of considerable importance to threatened or endangered wildlife.

Diagnosis of OP Poisoning.

Due to their relatively short life *in vivo* chemical analysis of OPs in tissue is of little value. Brain tissue, erythrocytes (RBCs), and plasma have all been used to analyze for AChE activity to monitor OP pesticide exposure or determine cause of mortality. Analysis of stomach contents can confirm OP pesticide poisoning in some cases. In birds and reptiles depression of brain AChE activity by 50% or more that of comparable control is considered indicative of OP pesticide poisoning. A similar value has not been established for amphibians and mammals.

In general brain ChE activity depressed by 20% or more that of comparable controls can be considered indicative of at least sublethal exposure. Though plasma and erythrocyte analysis is useful for serial sampling and where sacrifice of an animal is unpractical, intrepretation of AChE activity from blood components can be more difficult because little is known about their AChE's function and its relationship to brain AChE activity.

Appendix 1. A partial list of organophorphorus and carbamate pesticides and their primary use.

ORGANOPHOSPHORUS PESTICIDES

ACTIVE INGREDIENT	PRIMARY USE
acephate	Plant systemic insecticide
acethion	Insecticide
agvitor	Insecticide
Akton	Nonsystemic insecticide
acetoxon (acetofos)	Contact insecticide
amidithion	Plant systemic insecticide
amidothioate (Mitemate)	Miticide
amiprophos	Experimental herbicide
amiton	Plant systemic insecticide & acaricide
aphidan	Plant systemic insecticide & acaricide
apholate	Insect chemosterilant
Aspon (Chlordane)	Contact & fumigant insecticide
avenin	Plant systemic insecticide
azinophosmethyl (Gusathion)	Insecticide
azinophosethyl (Gusathion A)	Nonsystemic insecticide & acaricide
azothion	Insecticide & acaricide
azothoate	Experimental insecticide & acaricide
bensulide	Herbicide
Bomyl	Contact, residual insecticide & acaricide

bromophos	Nonsystemic insecticide & acaricide
butonate (tribuphon)	Insecticide
carbophenthion (mephocarp)	Insecticide & acaricide
CELA K-159	Animal systemic insecticide
Cerezin	Fungicide
chlofenvinphos	Insecticide
chlormephos	Nonsystemic soil insecticide
chlorphoxim	Insecticide
chlorpyrifos	Insecticide
Colep	Insecticide [1]
Conen (BEBP)	Fungicide
Coroxon	Synergist for anthelmintic phenothiazine
coumaphos	Animal systemic & contact insecticide, nematocide, synergist to other anthelminthic OPs
Coumithoate	Veterinary insecticide & miticide
cremart (S-2846)	Herbicide
crotoxyphos	Veterinary insecticide
crufomate	Animal systemic & contact insecticide & helminthicide
cyanofenphos	Insecticide
cyanophos	Insecticide
cyanothoate	Insecticide & acaricide
DAEP (Amiphos)	Plant systemic insecticde & acaricide

DDVP (dichlorvos)	Insecticide
DEF (butifos)	Defoliant
demephion	Plant sytemic insecticide
demeton	Plant systemic insecticide & acaricide
demeton methyl	Plant systemic insecticide
demeton-O-methyl sulfoxide (oxydemetonmethyl)	Insecticide
dialifor (dialifos)	Insecticide & acaricide
diazion	Insecticide & nematocide
Di-Capthon (dicapthon)	Insecticide
dichlofenthion	Nematocide & soil insecticide
dichlorvos	Contact & stomach insecticide & helminthicide
dicrotophos	Plant systemic insecticide
diisopropyl fluorophosphate (DFP, isofluorphage)	Insecticide with systemic activity
dimefox	Plant systemic insecticide
dimethoate (phosphamide)	Plant systemic insecticide & acaricide
dioxathion	Contact insecticide & acaricide
disulfoton	Plant systemic insecticide & acaricide
DMCP (Fujithion)	Insecticide
DMPA (Zytron)	Herbicide
Dowco 199	Fungicide
DSP (Kaya-ace)	Nematocide with some soil insecticide activity

edifenphos (EDDP)	Fungicide
endothion[2]	Plant systemic insecticide & acaricide
EPBP (S-Seven)	Soil insecticide
EPN	Insecticide & acaricide
ethion	Insecticide & acaricide
ethephon (Ethrel)	Plant growth regulator (promotes ripening, flowering disease & freezer resistance etc.)
Ethoate-methyl	Plant systemic insecticide
famphur	Animal systemic insecticide
fenitrothion	Insecticide & acaricide
fensulfothion	Soil insecticide & nematocide with contact and systemic activity
fenthion	General purpose contact & stomach insecticide with systemic activity
fonophos	Soil insecticide
formoathion	Systemic & contact insecticide & acaricide
formothion	Plant systemic insecticide
fosthietan	Soil insecticide & nematocide
Gophacide	Rodenticide
glyphosate	Herbicide
haloxon	Anthelminthic agent
hempa	Insect chemosterilant
IBP (Kitazin P)	Systemic fungicide & synergist to insecticide fenitrothion

Inezin (ESBP)	Fungicide
iodofenfos (jodfenfos)	Nonsystemic, contact, stomach insecticide
intration	Experimental plant systemic insecticide
isofenphos (Bay 92114)	Contact and stomach insecticide
isothioate	Plant systemic insecticide &acaricide
isoxathion	Insecticide
leptophos	Insecticide, experimental fungicide
malathion	Insecticide
mecarbam	Contact insecticide & acaricide with ovicidal activity & some plant systemic insecticidal activity
menazon	Plant systemic insecticide & seed & root tip dressing
mephosfolan	Plant systemic insecticide
merphos	Cotton defoliant
Metasystox S	Plant systemic insecticide
metepa	Insect chemosterilant
methamidophos	Insecticide & acaricide
methidathion	Nonsystemic insecticide & acaricide
methyl demeton (methyl systox)	Systemic insecticide
methyl parathion	Insecticide
Methyl Trithion	Insecticide & acaricide
mevinphos	Plant systemic & contact insecticide & acaricide

mipafox[3]	Plant systemic insecticide
monocrotophos	Plant systemic insecticide
morphothion	Plant systemic & contact insecticide
naled	Insecticide & acaricide
naphthalophos (Maretin)	Anthelminthic and insecticidal (Mediterranean fruit fly)
Narlene	Animal systemic insecticide & anthelminthic
Nellite	Plant sytemic insecticide
NIA 16388	Insecticide synergist
omethoate	Plant systemic insecticide
oxydemetonmethyl	Plant systemic insecticide
oxydisulfoton	Seed treatment insecticide
parathion (ethyl parathion)	Insecticide
parathion methyl	Insecticide
phenamiphos (fenamiphos)	Plant systemic nematocide
phenkapton (Phencapton)	Selective acaricide, effective against all life stages
phenthoate	Insecticide & acaricide
phorate (timet)	Plant systemic, vapor & contact insecticide
phosalone (bensophos)	Insecticide & acaricide
phosfolan	Plant systemic and soil insecticide
phosmet	Nonsystemic insecticide & acaricide, systemic against cattle grubs
phosphamidon	Plant systemic & contact insecticide

phostex	Miticide with good ovicide activity
phoxim	Contact & stomach insecticide
pirimiphos-ethyl	Insecticide & fungicide
pirimiphos-mehtyl	Contact & fumigant insecticide & acaricide
Potasan	Stomach insecticide
profenofos	Nonsystemic, contact & stomach insecticide & acaricide
propaphos (Kayaphos)	Insecticide
propetamphos	Contact insecticide with stomach activity
prophos (ethoprop, Mocap)	Nematocide-soil insecticide
prothoate	Plant systemic insecticide & mite ovicide
PTMD (Danifos)	Acaricide
pyrazophos	Systemic fungicide
Pytazothion	Plant systemic insecticide
pyridafenthion	Insecticide
pyrophosphate	Contact insecticide
quinalphos	Contact & stomach insecticide
R-3,828	Animal systemic insecticide
ronnel	Animal systemic insecticide, plant systemic insecticide & acaricide & public health midge larvae control
salithion	Insecticide
schradan	Plant systemic insecticide
sulfotep (sulfotepp)	Contact & fumigant insecticide & acaricide

sulprofos	Cotton insecticide especially against *Lepidoptera*
temephos (Abate)	Mosquito larvicide
Tepa (APO)	Insect chemosterilant
TEPP[4] (tetraethylpyrophosphate)	Contact & fumigant insecticide & acaricide
terbufos (Counter)	Soil insecticide
tetrachlorvinphos (stirofos)	Insecticide
Tetrathion	Plant systemic insecticide
thiometon	Plant systemic & contact insecticide & acaricide
thionazin	Plant systemic nematocide & soil insecticide
thio-TEPA	Insect chemosterilant
triamiphos	Fungicide with some systemic acaricide properties
triazophos (Hostathion)	Nonsystemic contact, stomach insecticide & acaricide
trichlorfon	Animal systemic insecticide & nematocide
trichloronate	Nonsystemic soil insecticide
Vamidothion	Plant systemic insecitcide

CARBAMATE INSECTICIDES

Carbaryl	Insecticide
Carbofuran	Soil insecticide
Propoxur	Household insecticide (cockroach)
Aldicarb	Plant suystemic insecticide (extremely tocix to mammals LD_{50} <1 ppm)

Methomyl	Insecticide with high ovicide activity

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[1] Developed by Monsanto Co. in 1962 but not marketed as of 1974.

[2] No longer available.

[3] Not used in practice because of delayed neurotoxicity.

[4] Under consideration for possible cancelation by USEPA 1983.

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